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Ambient Water Quality Criteria 2-Chlorophenol

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2-CHLOROPHENOL

Ambient Water Quality Criteria

Criteria and Standards Division
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CRITERION DOCUMENT

2-CHLOROPHENOL

CRITERIA

Aquatic Life

For 2-chlorophenol the criterion to protect freshwater aquatic life as derived using the guidelines is 60 µg/l as a 24-hour average and the concentration should not exceed 180 µg/l at any time.

For saltwater aquatic life, no criterion for 2-chlorophenol can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

Human Health

For the prevention of adverse effects due to the organoleptic properties of 2-chlorophenol in water, the criterion is 0.3 µg/l.

BIBLIOGRAPHIC INFORMATION

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Ambient Water Quality Criteria: 2-Chlorophenol.

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Section 304(a) of the Clean Water Act (33 U.S.C. 1314(a)), requires EPA to publish and periodically update water quality criteria. These criteria are to reflect the latest scientific knowledge on the identifiable effects of pollutants on public health and welfare, aquatic life, and recreation. This report presents water quality criteria for 2-chlorophenol. It presents concentration criteria for the protection of fresh water and saltwater aquatic life. It presents 'safe' concentrations for humans, and in the case of suspect or proven carcinogens, gives various levels of incremental cancer risk. A section 304(a) water quality criterion is a qualitative or quantitative estimate of the concentration of a water constituent or pollutant in ambient waters which, when not exceeded, will ensure a water quality sufficient to protect a specified water use. Under the Act a criterion is a scientific entity, based solely on data and scientific judgment. It does not reflect considerations of economic or technological feasibility nor is it a water quality standard and in itself h , ...

KEYWORDS: *Water quality, *Toxicology, *Water pollution effects(Humans), *Water pollution effects(Animals), *Toxic substances, *Phenol/chloro.

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2-CHLOROPHENOL

Introduction

2-Chlorophenol is a commercially produced chemical used entirely as an intermediate in the production of other chemicals. It represents a basic chemical feedstock for the manufacture of higher chlorophenols for such uses as fungicides, slimicides, bactericides, antiseptics, disinfectants, and wood and glue preservatives. 2-Chlorophenol is also used to form intermediates in the production of phenolic resins and has been utilized in a process for extracting sulfur and nitrogen compounds from coal.

2-Chlorophenol (ortho- oro-chlorophenol) is a substituted phenol having the empirical formula C_6H_5Cl. It has a molecular weight of 128.56, a density of 1.2573 at 25 degrees, and a vapor pressure of 1 mm Hg at 12.1 degrees C (Sax, 1975; Stecher, 1968). 2-Chlorophenol melts at 8.7 degrees C and exhibits a boiling point range of 175-176 degrees C (Rodd, 1954; Judson and Kilpatrick, 1949).

The spatial configuration and resonance effect of 2-chlorophenol may suppress the activity of the halogen atom via hydrogen bonding and partly account for the lower toxicity as compared to the 3- and 4-chlorophenol isomers (Haung and Gloyna, 1968).

In aqueous solution, 2-chlorophenol is slightly soluble (1,000 mg/l) at 25 degrees C and neutral pH (Henshaw, 1971; U.S. EOA, 1973). The log of the octanol/water partition coefficient for 2-chlorophenol is 2.16 (U.S. EPA, 1978). It is weakly acidic, possesses a pKa of 8.48 in water at

25 degrees C, and will dissociate in alkaline solutions (Judson and Kilpatrick, 1949; Pearce and Simpkins, 1968). The monovalent salts particularly are soluble in aqueous solutions and the degree of solubility is pH dependent.

Information concerning the presence and fate of 2-chlorophenol is incomplete or nonexistent. However, the generation of waste sources from the commercial production of 2-chlorophenol, its chemically derived products and the inadvertent synthesis of 2-chlorophenol due to chlorination of phenol in effluents and drinking water sources, may clearly indicate its importance in potential point source and non-point source water contamination.

The chlorination of phenol from dilute aqueous solutions (Aly, 1968; Barnhart and Campbell, 1972) and from sewage effluents (Jolly, 1973; Jolly, et al. 1975) have been demonstrated. However, no data regarding 2-chlorophenol concentrations in finished drinking water are available.

Microbial degradation of 2-chlorophenol under laboratory conditions has been reported. Studies on the metabolism of the herbicide, 2, 4-dichlorophenoxyacetate (2, 4-D), have demonstrated the dechlorination and aromatic ring degradation of 2-chlorophenol by an Arthrobacter species (Loos, et al. 1966). Nachtigall and Butler (1974) reported the complete oxidation of 2-chlorophenol by Pseudomonas sp. isolated from activated sludge. Although these laboratory studies suggested microbial oxidation as an important degradation route for 2-chlorophenol, sufficient data are not available to reach conclusions regarding the persistence of this compound in the environment.

Several studies have reported an odor threshold concentration of 2-chlorophenol in aqueous solutions. Other studies have demonstrated the property of 2-chlorophenol to produce undesirable taint in freshwater vertebrate species. Some work has further demonstrated that phenolic flavor can be acquired by fish flesh through contaminated fish food or by other animals eating contaminated fish.

Although 2-chlorophenol has been reported to be less toxic than the higher chlorophenols, its low odor threshold in water and its tainting properties are considered a potential threat to certain beneficial uses of water and the utilization of aquatic life as a food source.

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AQUATIC LIFE TOXICOLOGY*

FRESHWATER ORGANISMS

Introduction

Most of the toxicity data available for 2-chlorophenol has been acquired under static testing conditions without chemical measurements. Although this compound is quite soluble in water, one would expect some loss of the chemical through absorption by the animals and by the testing environment. This would result in a low estimate of the toxicity and thus require adjustment by use of the Guideline adjustment factors. The one flow-through test that was found does not provide sufficient data to suggest a deviation from the Guidelines. Only one chronic test has been run, and since no threshold level was attained, the data have limited value.

Although 2-chlorophenol does not appear to be extremely toxic to aquatic life, it has been shown to impair the flavor of the edible portions of fish at very low concentrations. This fact will therefore be used in deriving the criterion.

Acute Toxicity

Eleven acute tests were run, representing four species of fish (Table 1) and one invertebrate species (Table 2).

*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life [43 FR 21506 (May 18, 1978) and 43 FR 29028 (July 5, 1978)] and the Methodology Document in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are the calculations for deriving various measures to toxicity as described in the Guidelines.

Of these tests, only one was flow-through with measured concentrations. The adjusted LC50 values ranged from 2,185 µ/l for Daphnia magna (U.S. EPA, 1978) to 12,380 µg/l for fathead minnow (Phipps, et al. Manuscript). By species, the average adjusted concentration was 4,239 µg/l for Daphnia magna, 4,312 µg/l for bluegill, 6,763 µg/l for goldfish, 8,885 µg/l for fathead minnow, and 11,027 µg/l for guppy. For the limited information available, the range of species sensitivities appears to be relatively narrow.

Application of the Guideline adjustment and sensitivity factors to the fish acute tests results in a concentration of 1,800 µg/l. Since this concentration is lower than that for the flow-through test, it becomes the Final Fish Acute Value. The Final Invertebrate Acute Value is 180 µg/l, which becomes the Final Acute Value since it is lower than the Final Fish Acute Value.

The 96-hour LC50 values for chlorinated phenols and bluegills (U.S. EPA, 1978) are directly related to the degree of chlorination. These values decrease from 6,590 µg/l for 2-chlorophenol and 3,830 µg/l for 4-chlorophenol to 60 and 77 µg/l for pentachlorophenol. These data and those for intermediate chlorinated phenols can be correlated with the n-octanol/water partition coefficient with a correlation coefficient of 0.95. Data for other species do not correlate as well.

Chronic Toxicity

One fish chronic test was found (U.S. EPA, 1978), but no adverse effects were observed at the highest test concentration (3,900 µg/l). After adjustment for species sensitivity, the Final Fish Chronic Value is greater than 290 µg/l. There are no data available for chronic toxicity to invertebrate species.

Plant Effects

Only one test was conducted with plants (Huang and Gloyna, 1967), and the effect level (500,000 µg/l) indicates that plants may not be sensitive to 2-chlorophenol (Table 4).

Residues

A bioconcentration factor was found only for the bluegill (U.S. EPA, 1978). The test was run using ^{14}C-2-chlorophenol for 28 days at an exposure concentration of 9.2 µg/l, and the factor determined was 214 (Table 5). The depuration rate was rapid with a half-life of less than one day. Since no maximum permissible tissue level exists for this compound, no Residue Limited Toxicant Concentration can be calculated.

Miscellaneous

As stated in the introduction, 2-chlorophenol was found to impair the flavor of fish at lower concentrations than those at which it had a toxic effect (Henderson, et al. 1960 and Shumway and Palensky, 1973) (Table 6). In the former study, bluegills were exposed for periods of one to four weeks to 2,000 µg/l of 2-chlorophenol and various concentrations of a number of organic nitriles. A taste

panel of twelve members recorded their reaction to the cooked and coded fish samples. The only chemical that caused a definite panel reaction was 2-chlorophenol, which ranged from mild to quite severe nausea. No attempt was made to establish a level of exposure which would not cause flavor impairment. The other experiment (Shumway and Palensky, 1973), however, was designed to provide this information. In this study, rainbow trout were exposed for 48 hours to a range of concentrations of 2-chlorophenol, and a panel of fifteen judges scored the flavor of the flesh on an increasing impairment scale of 0 to 6. The results were then plotted against exposure concentrations and graphically interpreted to arrive at an estimate of the highest concentration which would not impair the flavor of the flesh. For 2-chlorophenol, this concentration was estimated to be 60 µg/l in the exposure water, and since this value is the lowest of all the data discussed, it becomes the Final Chronic Value.

CRITERION FORMULATION

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Freshwater - Aquatic Life

Summary of Available Data

The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 1,800 µg/l

Final Invertebrate Acute Value = 180 µg/l

Final Acute Value = 180 µg/l

Final Fish Chronic Value = greater than 290 µg/l

Final Invertebrate Chronic Value = not available

Final Plant Value = 500,000 µg/l

Residue Limited Toxicant Concentration = not available

Final Chronic Value = 60 µg/l for tainting of fish flesh

0.44 x Final Acute Value = 79 µg/l

The maximum concentration of 2-chlorophenol is the Final Acute Value of 180 µg/l and the 24-hour average concentration is the Final Chronic Value of 60 µg/l. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

CRITERION: For 2-chlorophenol the criterion to protect freshwater aquatic life as derived using the guidelines is 60 µg/l as a 24-hour average and the concentration should not exceed 180 µg/l at any time.

Table 1. Freshwater fish acute values for 2-chlorophenol

Organism	Bioassay Method*	Test Conc.**	Time (hrs.)	LC50 (ug/l)	Adjusted LC50 (ug/l)	Reference
Goldfish, Carassius auratus	S	U	96	12,370	6,763	Pickering & Henderson, 1966
Fathead minnow, Pimephales promelas	S	U	96	11,630	6,358	Pickering & Henderson, 1966
Fathead minnow, Pimephales promelas	S	U	96	14,480	7,916	Pickering & Henderson, 1966
Fathead minnow, Pimephales promelas	FT	M	96	12,380	12,380	Phipps, et al. Manuscript
Guppy, Poecilia reticulatus	S	U	96	20,170	11,027	Pickering & Henderson, 1966
Bluegill, Lepomis macrochirus	S	U	96	6,590	3,603	U.S. EPA, 1978
Bluegill (juvenile), Lepomis macrochirus	R	U	48	8,100	3,587	Lemmering & Burbank, 1960
Bluegill, Lepomis macrochirus	S	U	96	10,000	5,467	Pickering & Henderson, 1966
Bluegill (juvenile), Lepomis macrochirus	S	U	96	8,400	4,592	Henderson, et al. 1960

* S = static, FT = flow-through, R = renewal

** M = measured, U = unmeasured

Geometric mean of adjusted values = 7,211 ug/l $\quad \frac{7,211}{3.9} = 1,800$ ug/l

Lowest value from a flow-through test with measured concentrations = 12,380 ug/l

Table 2. Freshwater invertebrate acute values for 2-chlorophenol

Organism	Biomssay Method*	Test Conc.**	Time (hrs.)	LC50 (ug/l)	Adjusted LC50 (ug/l)	Reference
Cladoceran, Daphnia magna	S	U	48	7,430	6,293	Koppermsn, et al. 1974
Cladoceran, Daphnia magna	S	U	48	2,580	2,185	U.S. EPA, 1978

* S = static

** U = unmeasured

Geometric mean of adjusted values = 3,708 µg/l $\frac{3,708}{21}$ = 180 µg/l

Table 3. Freshwater fish chronic values for 2-chlorophenol (U.S. EPA, 1978)

Organism	Test	Limits (ug/l)	Chronic Value (ug/l)
Fathead minnow, *Pimephales promelas*	E-L	>3,900	>1,950

* E-L = embryo-larval

Geometric mean of chronic values = >1,950 µg/l $\quad \frac{>1,950}{6.7}$ = >290 µg/l

Lowest chronic value = >1,950 µg/l

Table 4. Freshwater plant effects for 2-chlorophenol (Huang & Gloyna, 1967)

Organism	Effect	Concentration (ug/l)
Alga, Chlorella pyrenoidosa	Reduction in chlorophyll in 72 hrs	500,000

Table 5. Freshwater residues for 2-chlorophenol (U.S. EPA, 1978)

Organism	Bioconcentration factor	Time (days)
Bluegill, *Lepomis macrochirus*	214	28

Table 6. Other freshwater data for 2-chlorophenol

Organism	Test Duration	Effect	Result ug/l	Reference
Rainbow trout, *Salmo gairdneri*	48 hrs	ETC*	60	Shumway & Palensky, 1973
Goldfish, *Carassius auratus*	8 hrs	42% mortality	31,100	Geredorff & Smith, 1940
Fathead minnow, *Pimephales promelas*	192 hrs	LC50	6,340	Phipps, et al. Manuscript
Bluegill, *Lepomis macrochirus*	1 wk	Flavor impairment	2,000	Henderson, et al. 1960

* ETC = the highest estimated concentration of material that will not impair the flavor of flesh of exposed fish.

SALTWATER ORGANISMS

Introduction

No data on the effects of 2-chlorophenol on saltwater aquatic life are available.

CRITERION FORMULATION

Saltwater - Aquatic Life

No saltwater criterion can be derived for 2-chlorophenol using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

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2-CHLOROPHENOL

Mammalian Toxicology and Human Health Effects

EXPOSURE

The potential for exposure of man to any synthetic chemical exists through any of several modes. These modes include: 1) exposure of industrial workers during synthesis, formulation, packaging or transport; 2) exposure of users of the product at either a commercial or retail level; 3) contact with residues or metabolites of the product as a result of using commodities or environments containing the material and; 4) contact with the chemical as a metabolite of some other product.

To understand route of entry of a chemical, one must first examine the sources and properties of the material.

2-Chlorophenol is a commercially produced chemical used as an intermediate in the production of other chemicals and it represents a basic chemical feedstock for manufacture of higher chlorophenols.

Direct chlorination leads to the formation of both 2- and 4-chlorophenols and the isomers are separated by fractional distillation since the difference in boiling points is greater than 40 degrees C. Most of the 2-chlorophenol used commercially in the U.S.A. is recovered as a by-product from the manufacture of 4-chlorophenol by direct chlorination of phenol.

The chlorination of phenol in aqueous solutions to form 2-chlorophenol and higher phenols has been demonstrated under conditions similar to those used for disinfection of waste water effluents and may represent a source of contamination (Aly, 1968; Barnhart and Campbell, 1972). Since chlorine and phenol do not normally occur in stoichiometric amounts, the concentrations of 2-chlorophenol actually produced in H_2O are likely to be lower than those found in this study (Weber, 1972). Higher levels of chlorination become increasingly less favored. 2-Chlorophenol has been synthesized from phenol and chlorine at concentrations as low as 10 and 20 mg/l, respectively, within 1 hour (Barnhart and Campbell, 1972). Other studies have demonstrated the formation of 2-chlorophenol (1.7 µg/l) and numerous other chlorinated compounds during the chlorination of sewage effluents and power plant cooling waters (Jolly, 1973; Jolly, et al. 1975).

Ingestion from Water

2-Chlorophenol may exist in the aquatic environment in the dissolved form, associated with suspended matter and bottom sediments, and absorbed in biological tissues. Metal salts of this compound have greater water solubility and if they are introduced or formed in situ they would exist primarily in the dissolved form. Chlorophenols being weak acids tend to ionize, depending upon the pH of the system. They are almost completely nonionized in aqueous solution with pH lower than 5 and become increasingly dissociated as the pH rises (Cserjesi, 1972).

There is no information on amounts of 2-chlorophenol present in finished water for human consumption.

In one study, industrial waste discharge was the principal point source of water pollution. During the manufacture of chlorophenols and 2,4-D there is chemical waste generated resulting from incomplete reaction of the starting reactants, by-product formation, and incomplete recovery of desired products. Thus, the wastes contain a mixture of chlorophenols and other compounds. Waste arising from the manufacture of phenoxyalkanoic herbicides showed amounts of 2-chlorophenol ranging from a trace to 6 percent (Sidwell, 1971) (Tables 1 and 2) of the total phenols and chlorophenols.

Other possible point sources are chemical spills and washing of containers or drums in which chlorophenols and 2,4-D are stored.

TABLE 1
Chlorophenols in Industrial Plant Waste

Date	25 January	3 March	21 April	28 May	27 August
Temp. ^{o}C	12	18	21	28.5	24
pH	7.5	7.6	7.4	7.4	7.0
Chlorophenols (mg/l)	68	118	125	112	74
Phenoxy Acids (mg/l)	167	183	241	235	199
Total Solids (mg/l)	6,960	40,100	76,320	104,860	11,000

Contamination of water with 2-chlorophenol may arise from 1) chlorination of phenol present in natural water and primary and secondary effluents of waste treatment plants (Burttschell, et al. 1959; Eisenhauer, 1964; Barnhart and Campbell, 1972), 2) direct addition of the chemicals or as contaminants or degradation products of 2,4-D used for aquatic weed control, and 3) wet and dry atmospheric fallout.

TABLE 2
Relative Chlorophenol Content of Industrial Waste

Date	25 January	3 March	21 April	28 May	27 August
Phenol Type	%	%	%	%	%
2-chloro	2.9	6.1	trace	trace	trace
phenol	3.4	6.2	1.7	24.8	trace
2,6 DCP	9.9	41.7	38.8	30.5	3.0
2,5 DCP	trace	6.2	1.7	trace	1.8
2,4 DCP	73.6	17.9	20.0	11.4	89.0
2,4,6 TCP	2.8	9.9	19.5	13.3	3.4
4-chloro	2.5	12.1	18.3	20.0	2.8
2,4,5 trichloro	4.7	trace	trace	trace	trace

No direct data were found to show actual measured concentrations of 2-chlorophenol in water courses, impoundments, wells or other human water supply sources.

Based on the relatively limited sources of water contamination by 2-chlorophenol as well as the demonstrated decomposition in many aquatic situations, water should be a minor route of ingestion of 2-chlorophenol.

2-Chlorophenol may be removed from water by several mechanisms. One study indicates that the dissipation of 2-chlorophenol (Ettinger and Ruchhoft, 1950) is largely microbiological. Persistence appears to be short but limnological factors such as oxygen deficiency may delay degradation (Aly and Faust, 1964). Microorganisms found in activated sludge and waste lagoons have been demonstrated to degrade the 2-chlorophenol rather readily (Sidwell, 1971; Nachtigall and Butler, 1974).

Ettinger and Ruchhoft (1950) found that low concentrations (1 mg/liter) of 2-chlorophenol added to a usual dilution of domestic sewage were not removed during periods of 20 to 30 days, presumably due to the absence of microorganisms capable of attacking the chemical. When a similar concentration was added to polluted river waters the compound dissipated in 15 to 23 days. Addition of a seed consisting of water from a previous persistence experiment increased significantly the removal of 2-chlorophenol. Apparently, the seed introduced some organisms already adapted to the chemical. This study also indicated that the removal of monochlorophenols requires the presence of an adapted microflora.

Ingols, et al. (1966) obtained data for the dechlorination of 2-chlorophenol and other monochlorophenols within 3 days when exposed to an activated sludge system (See Table 3).

TABLE 3

Degradation of Chlorophenols in Acclimated Activated Sludge[a,b].

Compound	Amount of ring degradation of compound		Development of chloride ion	
	%	days	%	days
2-chlorophenol	100	3	100	4
3-chlorophenol	100	2	100	3
4-chlorophenol	100	3	100	3
2,4-DCP	100	5	100	5
2,5-DCP	52	4	16	4
2,4,6-TCP	100	3	75	3
NaPCP	0	4	0	4
Dichloroquinone	100	1	50	3
2,5-DCP benzoquinone	30	1	0	1

[a]Source: Modified from Ingols, et al. (1966)
[b]Concentration of 100 mg/l

Primary treatment consists essentially of settling solids after screening off large materials. Settling may not remove 2-chlorophenol from water because these chemicals are sorbed poorly on particulate or suspended matter.

Secondary treatment involves the removal of organic matter from waste water by biological processes. Since 2-chlorophenol and 2,4-DCP are known to be easily biodegradable, secondary treatment should provide excellent removal of these chemicals.

Baird, et al. (1974) employing Warburg respiratory techniques demonstrated that biodegradation of 2-chlorophenol at 1 mg/liter in activated sludge was complete within 3 hours. Increasing the concentration to 100 mg/liter considerably reduced the rate of respiration such that only 20 percent was degraded in 6 hours. This is probably due to microbial toxicity from 2-chlorophenol at this concentration. In

a sludge not acclimated with high levels of 2-chlorophenol, certain amounts of the compound may be degraded initially while oxidative intermediates that appear subsequently could be toxic to the microbial population. This indicates that 2-chlorophenol may persist longer if waste containing high levels of the chemical is discharged to an unacclimated body of water due to direct or indirect toxic effects.

While a number of studies indicate rapid dissipation of 2-chlorophenol from waters by several mechanisms, human exposure cannot be fully evaluated unless studies are conducted measuring the 2-chlorophenol content in waters receiving wastes from point sources of chlorophenols or their precursors. Evidence of such studies was not found.

Ingestion from Food

Contamination of human foods with 2-chlorophenol could occur via soil, plants, animals or aquatic foods. In all cases, any contamination is probably indirect and a result primarily of use and subsequent metabolism of phenoxyalkanoic herbicides.

In 1971, U.S. farmers applied almost 16,000,000 kg of 2,4-D representing 15 percent of all organic herbicide usage (U.S.D.A., 1974).

Although 2-chlorophenol appears to be short-lived in soils, the data are inconclusive and factors affecting its persistence need further study. However, microbial degradation is the apparent major avenue of dissipation for these chemicals in soils. For 2,4-DCP, which is more likely to reach the soil system as a contaminant and degradation product of 2,4-D, its degradation under field conditions could

be faster than the herbicide. The role of microorganisms in the degradation of 2,4-D has been conclusively demonstrated (Loos, 1975) and under favorable conditions 2,4-D disappears from soils in about 30 days (Kearney, et al. 1969). Warm, moist, well-aerated soils with ample organic matter content promote the proliferation of microorganisms known to metabolize 2,4-D. One of the steps in the metabolic scheme may be 2-chlorophenol. Limited information indicates the biodegradability of 2-chlorophenol (Walker, 1954; Baird, et al. 1974). Several genera of bacteria isolated from soil are capable of metabolizing 2-chlorophenol. Pseudomonas sp., Nocardia sp., Mycobacterium coeliacum and Bacillus sp. were demonstrated to oxidize 2-chlorophenol to 3-chlorocatechol (Spokes and Walker, 1974). The fate of the catechol intermediate was elucidated from a study of the metabolism of 2,4-D by Pseudomonas sp. (Evans, et al. 1971). Using 2,4-D as sole carbon source for Pseudomonas strains isolated from soil, the herbicide was metabolized to 2,4-DCP, 2-chlorophenol, 3,5-dichlorocatechol and alpha-chloromuconate which was further metabolized to release Cl^- and unidentified metabolites. The appearance in culture of 2-chlorophenol suggests the non-oxidative elimination of chlorine from 2,4-DCP or possibly 2,4-D itself. The accumulation of alpha-chloromuconate is probably a further manifestation of this phenomenon since it is likely formed by enzymatic cleavage of 3-chlorocatechol, the latter, in turn, being derived from either 2-chlorophenol or 3,5-dichlorocatechol.

It is probable that the sorption behavior of 2-chlorophenol is similar to 2,4-D. In natural soil systems, sorp-

tion may not be extensive thereby favoring their downward movement in soil with water.

The persistence of 2-chlorophenol in soils was studied by Walker (1954) using the percolation technique. Solutions of 2-chlorophenol (1.0 g/4 l tap water) were allowed to percolate through 100 g of a Rothamsted soil (light clay with a pH of 6.8) and the disappearance of the initial and subsequent doses was measured. Two-thirds of the initial dose disappeared in 10 days. Disappearance of subsequent doses occurred approximately twice as rapidly as that of the first dose suggesting microbial participation. Further evidence of microbial decomposition was indicated by the more rapid disappearance of 2-chlorophenol in fresh than in sterilized soil within 7 days of percolation.

Furthermore, the participation of soil microorganisms in the dissipation of 2-chlorophenol and other chlorophenols was reported by Alexander and Aleem (1961) using suspensions of two silt loam soils. Metabolism of the chemicals was evidenced by more rapid disappearance of incremental additions of the compounds than initial enrichments. Also, inhibition of degradation occurred on addition of sodium azide, a toxic agent. 2-Chlorophenol disappeared rapidly in suspensions of Dunkirk and Mardin silt loams; disappearance was faster for the latter soil.

No information was found on the uptake, absorption, and translocation of 2-chlorophenol by plants. The movement of 2-chlorophenol can only be inferred from the few available studies of 2,4-dichlorophenol in plants and from the potential for 2-chlorophenol to occur as a metabolic intermediate in the degradation of 2,4-D.

The metabolism of 2-chlorophenol in vascular plants is not well studied. The only report found demonstrated that 2-chlorophenol may be inactivated by glycoside formation in plant tissue. It has been demonstrated that when certain non-naturally occurring chemicals are absorbed by various plants, glycoside formation takes place with the foreign chemical serving as the aglycon. Miller (1941) demonstrated that the metabolic fate of 2-chlorophenol in tomato plants included glycoside formation. Beta-o-chlorophenyl-gentiobioside (a glycoside of 2-chlorophenol) was isolated from the roots of these tomato plants. No evidence for the formation of this glycoside in shoots was found. The fate of this metabolic product of 2-chlorophenol in plants is not known and warrants further investigation.

Domestic animals including poultry could ingest feeds containing pesticides or drink water contaminated directly with 2-chlorophenol and 2,4-DCP. Although some studies indicate the appearance and distribution of 2,4-DCP in tissues of animals and poultry fed with 2,4-D and nemacide (O(2,4-Dichlorophenol) 0,0-Diethyl Phosphorothioate)) (Clark, et al. 1975; Sherman,et al. 1972), in none of the studies was there evidence cited to indicate residues of 2-chlorophenol. Furthermore, Bjerke, et al. (1972) reported no contamination of milk and cream from cows dosed with 2,4-D (100 to 1,000 mg/kg) treated foods.

A bioconcentration factor (BCF) relates the concentration of a chemical in water to the concentration in aquatic organisms, but BCF's are not available for the edible portions

of all four major groups of aquatic organisms consumed in the United States. Since data indicate that the BCF for lipid-soluble compounds is proportional to percent lipids, BCF's can be adjusted to edible portions using data on percent lipids and the amounts of various species consumed by Americans. A recent survey on fish and shellfish consumption in the United States (Cordle, et al. 1978) found that the per capita consumption is 18.7 g/day. From the data on the 19 major species identified in the survey and data on the fat content of the edible portion of these species (Sidwell, et al. 1974), the relative consumption of the four major groups and the weighted average percent lipids for each group can be calculated:

Group	Consumption (Percent)	Weighted Average Percent Lipids
Freshwater fishes	12	4.8
Saltwater fishes	61	2.3
Saltwater molluscs	9	1.2
Saltwater decapods	18	1.2

Using the percentages for consumption and lipids for each of these groups, the weighted average percent lipids is 2.3 for consumed fish and shellfish.

A measured steady-state bioconcentration factor of 214 was obtained for 2-chlorophenol using bluegills containing about 1 percent lipids (U.S. EPA, 1978). An adjustment factor of 2.3/1.0 = 2.3 can be used to adjust the measured BCF from the 1.0 percent lipids of the bluegill to the 2.3 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average bioconcentra-

tion factor for 2-chlorophenol and the edible portion of all aquatic organisms consumed by Americans is calculated to be 214 x 2.3 = 490.

Inhalation

The dispersal and distribution of 2-chlorophenol in air has apparently not been studied. One potential source of environmental pollution by 2-chlorophenol, however, is the manufacture of 2,4-D herbicides. Secondly, since 2-chlorophenol is volatile (1 mm Hg at 12 degrees C) any 2-chlorophenol generated as a decomposition product of 2,4-D could be subject to general environmental dispersal wherever 2,4-D is applied. A third possibility for inhalation exposure could be burning of containers, trash or plant material contaminated with 2-chlorophenol. No data addressing the monitoring of air or workplace environments for 2-chlorophenol has been found. Therefore, the potential sources for inhalation exposure remain speculative. However, the relative potential for exposure should be considered.

Because of the processes employed in manufacture of 2-chlorophenol as well as its volatility, the most probable source of inhalation exposure to 2-chlorophenol would occur in manufacturing plants producing 2-chlorophenol or possibly 2,4-D. In addition, the separation of 2-chlorophenol from 2,4-dichlorophenol involves fractional distillation which, if not done with regard to worker safety, could result in exposure by inhalation. The potential for airborne exposure to 2-chlorophenol in the general environment as a result of point source pollution has not been reported.

Although inhalation exposure to 2-chlorophenol associated with related products in general use (2,4-D herbicide) does not seem likely, no data to verify atmospheric 2-chlorophenol presence or absence under such conditions have been found. Potential for such exposure seems quite low for several reasons. First, the principal general environmental source of 2-chlorophenol would be 2,4-D or its decomposition products. Since there is little evidence of 2-chlorophenol occurring as a permanent soil or plant metabolite of 2,4-D then the amount available to be volatilized would be either limited or absent. Secondly, any 2-chlorophenol which might be formed in soil or water is rapidly degraded by microorganisms (see section on Ingestion) while 2-chlorophenol in plants is inactivated as a glycoside (Miller, 1941).

A third potential route of exposure, burning of chlorophenol-containing products, has not been studied. Incineration of phenoxy herbicides should include investigations of potential formation and/or dispersal of 2-chlorophenol as well as other chlorophenols.

While direct studies of potential or actual exposure to 2-chlorophenol have not been found, after considering the nature of production, uses and persistence of 2-chlorophenol, inhalation exposure of the general population does not seem a significant threat.

Dermal

2-Chlorophenol dermal absorption data have not been found. Since the compound is lipid soluble and likely to be poorly ionized at environmental pH (Farquaharson, et al. 1958) it should be readily absorbed through intact skin.

The dermal absorption and resultant body burden should be studied.

As indicated for inhalation exposure, the only potentially significant dermal exposure to 2-chlorophenol would be in the manufacture or handling of 2-chlorophenol or products which contain it. Ordinary and accepted methods of skin protection would be expected to prevent dermal exposure to 2-chlorophenol. Dermal exposure to 2-chlorophenol from other sources (soil, water, plant metabolites of 2,4-D) is considered insignificant for the same reasons as stated in the section on inhalation.

PHARMACOKINETICS

Absorption

Direct data on the absorption of 2-chlorophenol by man or experimental animals have not been found. Chlorophenol compounds are generally considered readily absorbed and this would be expected from their high lipid solubility and low degree of ionization at physiological pH (Doedens, 1963; Farquharson, et al. 1958). Although skin irritation and dermal absorption are reported as characteristic of monochlorophenols, direct quantitative data concerning 2-chlorophenol irritant potential have not been found. Toxicity studies to be discussed later indicate that 2-chlorophenol can be absorbed and can result in toxicosis. However, quantitative data for 2-chlorophenol absorption by various routes have not been found.

2-Chlorophenol may occur in mammals indirectly as a metabolite of other compounds. Exposure of rabbits to chlorobenzene has resulted in formation of 2-chlorophenol (Lindsay-Smith, et al. 1972).

In addition, Selander, et al. (1975) reported the conversion of chlorobenzene to a mixture of monochlorophenols in perfused rat liver. Apparently, three different enzyme systems catalyze the conversion of chlorobenzene to 2-, 3-, and 4-chlorophenols.

Investigation of 2,4-D metabolism in mammals (Clark, et al. 1975) has not indicated 2-chlorophenol to be a metabolite of such exposure, while 2,4-DCP is considered the major metabolite.

Distribution

Direct information about the distribution and transport of 2-chlorophenol is not available. However, at least two reports (Spencer and Williams, 1950; Von Oettingen, 1949) on the rabbit and dog respectively, indicate urinary excretion of 2-chlorophenol. Furthermore, since metabolites of 2-chlorophenols are identified as glucuronide and sulfate conjugates, it is possible that the liver might contain proportionally large amounts of 2-chlorophenol. Two reports concerning lesions induced by 2-chlorophenol (Patty, 1963; Bubnov, et al. 1969) indicate changes in liver and kidney thus visually confirming the renal and hepatic distribution. No information about hepatic excretion nor any indication of an enterohepatic cycle was found. While compounds of high lipophilia (which would include 2-chlorophenol) are often considered to accumulate in adipose tissue, no information in this regard was found for 2-chlorophenol. In fact, related compounds (2,4-Dichlorophenol, Pentachlorophenol) are considered to have relatively short half-lives (Clark,

et al. 1975; Osweiler, et al. 1977). Whether this is true for 2-chlorophenol remains to be established. Since animals dosed with 2-chlorophenol display convulsive activity within several minutes of exposure (Farquharson, 1958; Angel and Rogers, 1972) it can be assumed the compound traverses the blood brain barrier and is distributed in part in the CNS. The concentrations of 2-chlorophenol in brain and other organs or tissues during toxicosis remain to be determined.

Metabolism

The metabolism of 2-chlorophenols in man is not known. In experimental animals Von Oettingen (1949) cites work by Karpow (1893) showing that dogs excreted 87 percent of administered 2-chlorophenol as conjugates with sulfate and glucuronic acid. The rabbit also apparently conjugates the 2-chlorophenol derived from chlorobenzene exposure (Lindsay-Smith, et al. 1972) by formation of sulfate and glucuronide conjugates. However, 2-chlorophenol was reported as a minor metabolite of chlorobenzene in the rabbit (see Table 4). Furthermore, only a minor portion of chlorophenols formed were monochlorophenols and less than 6 percent of free and metabolized chlorophenols were the ortho- or 2-chlorophenol isomer.

Selander, et al. (1975) demonstrated that chlorobenzene is converted to o-, m- and p- chlorophenols in perfused rat livers as well as by noncellular systems including microsomes, post-mitochondrial supernatant, and reconstituted soluble hemoprotein-monoxygenase systems. Pretreatment

TABLE 4
Lethal Doses of 2-Chlorophenol for Experimental Animals[a]
(LD-50 in mg/kg)

Animal	Route of administration	LD-50	Author
		2-Chlorophenol	
Rat	Oral	670	Deichmann, 1943
Rat	Subcutaneous	950	Deichmann, 1943
Albino rat	Intraperitoneal	230	Farquharson, et al. 1958
Rabbit	Subcutaneous	950[b]	Christensen & Luginbyhl, 1975
Rabbit	Intravenous	120[b]	Von Oettingen, 1940 citing Kuroda (1926)
Mouse	Oral	670	Bubnov, et al. 1969
Guinea pig	Subcutaneous	800[b]	Christensen & Luginbyhl, 1975
Blue fox	Oral	440	Bubnov, et al. 1969
Unknown mammal	Oral	440	Christensen & Luginbyhl, 1975

[a]Source: Assembled from several sources listed in body of the table.
[b]MLD (minimum lethal dose) values.

with the inducing agents 3-methylcholanthrene and phenobarbital increased the formation of chlorophenols while carbon monoxide and SKF 525a inhibited formation of o-, m-, and p-chlorophenols. The approximate ratios in vivo for formation of o-, m-, and p-chlorophenol were 1:2:4 respectively. Thus, formation of 2-chlorophenol via metabolism from chlorobenzene dose not appear a significant or major route of exposure. While it is possible that 2-chlorophenol could form in man or animals as a result of exposure to phenoyxacetic acid herbicides, there are no data to support this conjecture. In fact, Clark, et al. (1975) in studying the metabolism of phenoxy herbicides reported the major metabolite to be 2,4-DCP and did not mention detection of 2-chlorophenol.

Based on experimental work in two species (dogs and rabbits) it appears that mammalian metabolism of 2-chlorophenol follows the expected route for phenol metabolism, i.e. formation of conjugates of glucuronides and sulfates with detection of these metabolites primarily in the urine.

Excretion

Studies of the excretion route or rate for 2-chlorophenol in man were not found. Von Oettingen (1949) reviewed the data of Karpow (1893) in which dogs given 2-chlorophenol excreted 87 percent of the compound in urine as sulfate and glucuronide conjugates. However, data were not developed from which rate of excretion or half-life could be calculated. Lindsay-Smith, et al. (1972) identified phenolic metabolites in rabbits urine after administration of chlorobenzene. Of the free and conjugated forms of chlorophenols in rabbit urine, less than 6 percent were present as 2-chlorophenol.

No data have been found concerning measurement of tissue residues of 2-chlorophenol either from direct administration or by formation as a metabolite of other compounds, nor have sufficient data accumulated to allow calculation of a half-life for 2-chlorophenol.

EFFECTS

Acute, Subacute and Chronic Toxicity

The acute toxicity of 2-chlorophenol has been studied in a variety of organisms. The compound is considered to be an oxidative uncoupler (Mitsuda, et al. 1963) and a convulsant poison (Farquharson, et al. 1958; Angel and Rogers, 1972). No reports of the subacute or chronic toxicity of 2-chlorophenol have been found. This represents

a serious data gap in the toxicologic evaluation of 2-chlorophenol.

Mammalian toxicity of 2-chlorophenol has not been well studied. There are no reports of human or domestic animal toxicoses from accidental or intentional exposure to 2-chlorophenol. Furthermore, there is no evidence linking 2-chlorophenol exposure in industrial workers to the chloracne often associated with higher chlorophenols. Nor is there evidence to suggest that the toxic dioxins are contaminants of or are formed from 2-chlorophenol. No reports of chronic toxicity studies in domestic or laboratory animals have been found.

Doedens (1963) briefly characterizes the toxicity of 2-chlorophenol as being "likely" to be corrosive and irritating to eyes and skin. However, specific data or effects due to 2-chlorophenol were not presented. The only toxicologic data from which evaluation of 2-chlorophenol can be made result from a relatively few acute toxicologic studies in laboratory mammals (see Table 4). It may be seen by inspection of Table 4 that the subcutaneous minimum lethal dose (MLD) of 2-chlorophenol in the rabbit (800 to 950 mg/kg) is approximately 8 times that of the intravenous MLD implying that the subcutaneous route retards bioavailability of 2-chlorophenol. At a physiological pH of 7.4, 2-chlorophenol will be approximately 5 percent ionized (Farquharson, et al. 1958) which would not account for a lessening of toxicity by the subcutaneous route.

The oral LD50 indicates that 2-chlorophenol is more toxic by the oral than the subcutaneous route. At relatively acid pH (e.g. stomach or intestine) the pKa of 2-chlorophenol (8.65) would allow for a highly unionized state with ready absorption from the stomach or upper intestine. This effect could explain the greater toxicity of 2-chlorophenol orally.

Among the various species tested by the same route there is surprising similarity of acute toxicity. This would imply that initial absorption, metabolism, detoxification and organs affected are quite similar among various species. It would be expected then that chronic toxicity would vary according to ability of a species to metabolize, inactivate and excrete 2-chlorophenol on a long term basis. Unfortunately, studies of long term or chronic effects have not been reported.

Signs of 2-chlorophenol intoxication in rats are similar whether the compound is administered subcutaneously, intraperitoneally, or orally. The toxicological picture includes restlessness and increased rate of respiration within a few minutes following administration. Somewhat later motor weakness develops and tremors and convulsions induced by noise or touch occur. Eventually, dyspnea and the appearance of coma result and continue until death (Farquharson, et al. 1958). Following fatal poisoning, lesions in the rat include marked kidney injury, red blood cell casts in the tubules, fatty infiltration in the liver

and hemorrhages in the intestine (Patty, 1963). Bubnov, et al. (1969) report a similar pathological picture in the blue fox and the mouse. At lethal concentrations, 2-chlorophenol caused fatty degeneration of the liver, renal granular dystrophy and necrosis of the stomach and intestinal mucose. These signs are very similar to acute phenol toxicosis (Patty, 1963).

The convulsive action of 2-chlorophenol in mice was studied by Angel and Rogers (1972). Following intraperitoneal administration of 2-chlorophenol, a rapid onset of convulsions was noted. A simple exponential decay of the convulsive effect was noted which the authors speculated could have been a result of removal from the CNS by a simple chemical reaction. However, no information directly addressing this point is available.

Farquharson, et al. (1958) state that as phenol is progressively chlorinated the molar toxicity shows a tendency to increase when pK value falls below 7. Furthermore, convulsions are the most characteristic effect of chlorophenols with pK values of 8.65 or higher. Thus, it may be that convulsions are in some way associated with undissociated molecules. No studies were found which attempted to evaluate the passage of chlorophenols with different pK values across the blood brain barrier.

Synergism and/or Antagonism

Reports of studies directly assessing the synergism or antagonism of 2-chlorophenol by other compounds were not found. Since 2-chlorophenol is a weak oxidative uncoupler (Mitsuda, et al. 1963) it may be expected that concomitant exposure to other uncouplers (e.g. pentachlorophenol, dinitrophenol) would enhance that effect. In addition, exposure to chlorinated hydrocarbon insecticides with their characteristic convulsant activity could be at least additive in that regard.

Any agent causing liver damage sufficient to decrease the conjugation of 2-chlorophenol with glucuronide or sulfate could conceivably alter the excretion and/or toxicity of the parent compound. However, there are no specific studies to reflect such an effect. It is only speculation that the general tendency of conjugation to render a compound less toxic and more amenable to excretion would also operate in the case of 2-chlorophenol.

Teratogencity

No studies were found which addressed the teratogenicity of 2-chlorophenol.

Mutagenicity

No studies were found which addressed the mutagenicity of 2-chlorophenol.

Carcinogenicity

The report of Boutwell and Bosch (1959) is the only one found dealing with tumorigenicity of 2-chlorophenol. Repeated application of phenol and some substituted phenols has been reported to promote skin tumors in mice after a single initiating dose of dimethylbenzanthracene (DMBA). Papillomas have developed in mice (not exposed to DMBA) treated with phenol alone. In the studies of Boutwell and Bosch (1959) two trials included evaluation of 2-chlorophenol. In one of these, 25 μl of a 20 percent solution of 2-chlorophenol was applied twice weekly to female Sutter mice 2-3 months of age for 15 weeks. This application followed an initiating dose of 0.3 percent DMBA in benzene. Tumorigenic response was measured as follows:

1) The percentage of surviving mice bearing one or more papilloma was ascertained.
2) The total number of papillomas on <u>all</u> surviving mice was counted and divided by the number of survivors to give the average number of papillomas per mouse.
3) The number of mice bearing malignant tumors was determined.

Results of the promoter trial with 2-chlorophenol are presented in Table 5. Related promoter experiments with phenol as well as the benzene control are included for comparative purposes. Based on the data, the authors concluded that the promoting activity of 2-chlorophenol is similar to that of phenol.

TABLE 5

Appearance of Skin Tumors in Mice Treated Cutaneously with Phenols Following a Cutaneous Dose of 0.3% Dimethyl Benzanthracene (DMBA) in Acetone[a].

Treatments[b]	Time animals examined (week)	No. of mice (survivors/total)	Survivors with papilloma (%)	Average papillomas per survivor	Survivors with epithelial carcinoma (%)
Benzene control	12	12/12	0	0	0
10% phenol in benzene. No DMBA	20	24/30	33	0.62	13
20% phenol in acetone	12	21/24	58	-	5
20% phenol in benzene	24	10/33	100	3.20	20
20% 2-chlorophenol in benzene	15	31/35	61	1.48	10
20% 2-chlorophenol in dioxane. No DMBA	12	28/30	46	0.64	0

[a] Source: Modified from Boutwell and Bosch, 1959.
[b] All received DMBA except where stated.

In a second experiment, Boutwell and Bosch (1959) administered 20 percent 2-chlorophenol in dioxane as before for 12 weeks, but without an initiator. Results of this trial are also in Table 5. In both trials 2-chlorophenol was associated with a high incidence of papillomas. When DMBA was used as an initiator 10 percent of the survivors developed carcinoma at the skin site of application. When 2-chlorophenol alone was used there was no carcinogenic response.

Since the study was designed primarily to detect promoting activity, the effect of 2-chlorophenol as a primary carcinogen is not well defined. The study uses dermal applications of a phenolic compound at 20 percent concentration in organic solvents. The concentration is high enough that hair follicles and sebaceous glands are destroyed. The papillomatous response observed may have developed in response to chemical and/or physical damage from application of an irritant compound. Even with this harsh treatment no malignant neoplasia are observed except when DMBA had been used as an initiator. The only neoplasia observed was at the site of the direct application. This study does not evaluate systemic carcinogenesis and the route of administration is not appropriate to the model for carcinogenic risk assessment. The route of administration (dermal) has no established relationship to oral exposure.

An odor threshold for 2-chlorophenol in water has been reported by at least two investigators. Hoak (1977) determined the odor threshold of 2-chlorophenol to be 0.33 µg/l at 30°C and 2.5 µg/l at 60°C. Determination of the detect-

able odor was made by a panel of from two to four people comparing flasks of test water to a flask of odor-free water. The lowest concentration detected by any panel member was taken as the odor threshold. Hoak speculated that odor should be expected to become more noticeable as temperature increases; however, in evaluating a series of chlorophenols and cresols, it was found that some compounds had higher odor thresholds at 30°C while others were higher at 60°C. Burtschell, et al. (1959) made dilutions of chlorophenol in carbon-filtered tap water and used a panel of from four to six people to evaluate odor. Tests were carried out at room temperature which the investigator estimated to be 25°C. If a panel member's response was doubtful, the sample was considered negative. The geometric mean of the panel responses was used as the odor threshold. The data presented do not indicate a range of responses, but owing to the variability inherent to such procedures, it seems reasonable that the odor threshold for some people in the Burtschell group estimated downward toward the 0.33 µg/l figure of Hoak.

Studies on the impairment of fish flavor by 2-chlorophenol have also been reported. Henderson, et al. (1961) found that a concentration of 2,000 µg/l caused impaired flavor of bluegill sunfish after a 28-day static, renewal exposure. Only one concentration was tested, so no dose-related threshold was determined. Shumway and Palensky (1973) found 60 µg/l to be an estimated threshold concentration during a 48-hour flow-through exposure to rainbow trout.

Shultz (1961) determined that 15 µg/l affected the flavor of carp after a 3-day flow-through exposure. Boetius (1954) studied the flavor impairment of eels and oysters (species unspecified) in static systems and found flavor impairment in brackish water at 0.125 µg/l after 11 days for eels and 4 days for oysters. Methodology for determining flavor impairment was particularly lacking in the Boetius paper. Because of the subjectivity of flavor impairment, test methodology (especially in the selection of and evaluation by the test panel) is particularly important for the critical evaluation of a flavor impairment study.

CRITERION FORMULATION

Existing Guidlines and Standards

As far as can be determined, no standards or guidelines exist for 2-chlorophenol.

Current Levels of Exposure

Overall, exposure of the general population to 2-chlorophenol would be most likely in the form of consumption of phenolic-containing chlorinated drinking water. This would limit exposure primarily to water supplies contaminated by a point source of 2-chlorophenol. Such sources should be relatively easy to identify and monitor since analytical techniques for 2-chlorophenol are available. Apparently, such monitoring is not generally being done.

Since 2-chlorophenol is not a universally reported metabolite of 2,4-D, exposure of the general population via use of 2,4-D is only speculative. If small amounts of 2-chlorophenol are formed and gain access to ground water or the soil, it is not expected to persist in view of its ready susceptibility to microbial attack.

Inhalation or dermal exposure to the general population is not expected to be a significant part of any total 2-chlorophenol exposure.

For industrial workers manufacturing or handling 2-chlorophenol, inhalation exposure could be the greatest threat and the hardest to control. Dermal exposure in such instances should be negligible if sensible and accepted industrial hygiene practices are followed.

Due to the lack of monitoring data or human body burden values, human exposure cannot be determined.

Special Groups at Risk

The only special group expected to be at risk for high exposure to 2-chlorophenol are industrial workers involved in manufacturing or handling of 2-chlorophenol. No data were found to relate exposure or body burden to conditions of contact with 2-chlorophenol.

Basis and Derivation of Criterion

Insufficient data exist to indicate that 2-chlorophenol is a carcinogenic agent. The only study performed (Boutwell and Bosch, 1959) was designed to detect the promoting activity of 2-chlorophenol with dimethylbenzanthracene initiated tumors. (Under certain environmental conditions, 2-chlorophenol may produce a small amount of dibenzo-p-dioxin, which is an unsubstituted analog of chlorinated dibenzo-p-dioxins. The recent NCI bioassay report of possible carcinogenicity of dibenzo-p-dioxin[b]/ has concluded that dibenzo-p-dioxin was not carcinogenic for Osborne-Mendel rats or B6C3F1 mice.) In fact, insufficient health effects data exist on any chronic or acute effect of 2-chlorophenol. In view of this, the recommended criterion is based on organoleptic effects.

The data of the two investigators evaluating the odor of 2-chlorophenol in drinking water indicated that a low concentration is capable of causing discernible odor. Neither worker indicated if the threshold odor concentration made the water unacceptable for consumption. These studies coupled with flavor impairment studies suggest that the selection of 0.3 µg/l 2-chlorophenol would be sufficient for the prevention of adverse organoleptic effects in water. It should

be emphasized that this is a criterion based on aesthetic rather than health effects. Data on human health effects need to be developed as a more substantial basis for setting a criterion for the protection of human health.

Thus, based on the prevention of adverse organoleptic effects, the interim criterion recommended for 2-chlorophenol is 0.3 μg/l.

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